sufficient food to appease his appetite. In the early stage of severe cases it is always difficult to give more than 3000 calories a day; in the steep-curve period and in convalescence, patients take readily from 4000 to 6000 calories a day.

3. If any article of food causes persistent disturbances of digestion, the quantity given should be diminished, or the food should be stopped; otherwise the object of the high calory diet, the maintenance of the patient's nutrition, is defeated. If a patient cannot take all the food he requires, he should be given all he can digest and absorb.

4. Carbohydrates should furnish the greater part of the energy of the diet. The daily protein ration should not be below 62 grams, nor greatly exceed 94 grams. Clinical evidence indicates that a diet rich in fat may be taken by typhoid fever patients with benefit. Fat has furnished in some cases from one-third to one-half of the total energy of the food.

5. In the cases studied, the high calory diet has apparently modified the course of the disease, shortened convalescence, and reduced the mortality.

Finally, I wish to express my indebtedness and my thanks to Dr. Dana for the privilege of studying cases in his service, to Drs. W. Murray Kerr, T. J. Kearns, W. E. Lowthian, T. R. Pooley, Jr., Harold De Wolf, and William Tomkins, House Physicians in different years, for valued assistance, and to Miss Mary E. Sheehan, Head Nurse in Ward A1, for many practical suggestions and for her untiring aid in carrying out the administration of the diet.

MULTIPLE CONGENITAL HEMANGIO-ENDOTHELIOMAS OF THE LIVER.

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(From the McManes Laboratory of Pathology, University of Pennsylvania.)

The findings at autopsy in this case from the medical service of Dr. Le Boutillier, at St. Christopher's Hospital, Philadelphia, are so uncommon that we feel justified in placing the case on record. The subject was a female infant, aged ten weeks, one of twins, breast-fed. From birth a distention of the abdomen was noted which constantly and rapidly increased; the infant at the same time became each day weaker. On examination, the liver was found greatly enlarged, extending to the umbilicus and possessing a palpably nodular surface. Otherwise physical examination proved negative. The urine examination and the Wassermann reaction were negative. The temperature was slightly subnormal. In the hospital the child continued to become weaker, and without further symptoms died.

Autopsy was performed on November 3, 1910, a few hours after death, by Drs. Veeder and Austin. The abstract of the protocol from the records of the McMane's Laboratory of Pathology is as follows:

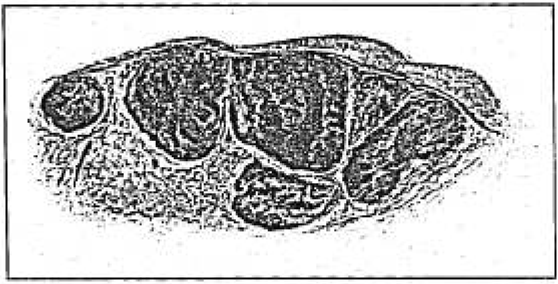
Body of a fairly well-nourished female infant. Slight postmortem rigidity and cadaveric lividity present. Abdomen markedly distended.

Abdomen. Primary incision showed a thin panniculus adiposus, no bleeding. Peritoneum smooth and glistening. Very slight excess of thin, clear fluid present in the abdominal cavity. No adhesions.

Liver. The liver was greatly enlarged, extending down as far as, and to the left of, the umbilicus, weighed 595 grams, and measured 17 x 9.5 x 6 cm. Its surface showed numerous dark, purplish nodules standing out prominently from the smooth peritoneal covering. These nodules varied in size from that of a pea to that of a horse-chestnut, were slightly umbilicated, and gave the liver an irregular, knotty appearance. Over the dome of the liver 19 of these nodules were counted, and on the under surface, 23. A typical nodule (2 cm. in diameter) had a dark red centre 1.4 cm. in diameter, surrounded by a white translucent zone 1 mm. in width. Outside of this was another dark zone from 1 to 3 mm. in width, apparently due to congestion, with radiations extending from it into the surrounding liver tissue. The nodules were distinctly softer than the surrounding liver substance, and were of about the consistency of brain. On section nodules were found throughout the substance of the liver. They showed, on cut surface, very dark, bluish-red centres, surrounded by a zone of pale white tissue from which branching processes extended inward toward the centre of the nodule. The liver tissue between the nodules was firm, quite yellow, somewhat congested, and showed the lobules faintly demarcated.

Sections of the tumor nodules and liver tissue were embedded and cut in paraffin, and stained with hematoxylin and eosin for microscopic examination.

Microscopic Description. The centres of the tumor nodules are composed of dilated blood spaces, irregularly arranged between numerous narrow trabeculæ of fibrous tissue and atrophic liver cells. The endothelial cells covering the trabeculæ are much more numerous than in the wall of a normal vessel, and in places have proliferated to form a double layer. They have large oval and somewhat vesicular nuclei, and occasionally show mitotic figures. The blood spaces are densely packed with erythrocytes and a few leukocytes. Around each nodule is an irregular capsule of compressed and fibrosed liver tissue with atrophic and vacuolated liver cells. The capsule of Glisson persists, and shows considerable overgrowth of fibrous tissue, particularly about the bile ducts. At some points the dilated blood channels are apparently dissecting their way into the liver substance, and here a transition can be seen from the normal to the atrophic liver cells. In some places the tumor tissue comes into apposition with the capsule of the liver, while in others a moderate layer of degenerated liver cells and fibrous tissue intervenes. The liver tissue uninvolved by the tumor shows advanced fatty change, the cells being occupied by large vacuoles. The protoplasm stains poorly and is granular. The connective tissue of the parts of the liver uninvolved by the tumor does not show hyperplasia.



Cut surface of liver.

The heart, lungs, thymus, spleen, kidneys, pancreas, adrenals, and abdominal lymph nodes were examined both grossly and microscopically, and no lesions of any moment were found. The only other pathological findings, and apparently they bear no relation to the tumors, were two small ulcerations of the duodenum situated just beyond the pylorus, about 4 mm. in diameter, extending through the mucosa.

From the histological picture the diagnosis of cavernous hemangiomas is evident. The dark centres of the nodules, seen grossly, correspond to the large collections of blood between the newly formed and growing bloodvessel walls. The white encapsulating area corresponds to the surrounding band of fibrous tissue with its atrophic liver cells. The septa passing toward the centre of the nodules from the periphery are bands of overgrown fibrous tissue along the bile ducts and larger bloodvessels.

Angiomas of the liver are not uncommon, but have rarely been reported in infancy or childhood. Primary sarcoma and carcinoma are far more common in childhood, as is shown by the large number of cases which Steffan[1] was able to collect. In searching the literature we have frequently found the statement that angiomas occur often in childhood, but it is seldom that references are given or cases cited, and we are, therefore, inclined to attribute this statement to the assumption by the authors of a congenital origin for the angiomas found in adults rather than to the discovery of the tumor during early life.

Cases of solitary angioma in infants have been reported by Steffan,[2] Michailow,[3] Kaufman,[4] and others, in all, however, less than a dozen.

The number of cases reported of multiple hemangiomas of the liver in children is even smaller. We have, in fact, been able to find but 3. One of these, reported by Chervinsky,[5] in an infant aged six months, is in many respects identical with ours. It was briefly as follows:

A child, born at term, of healthy parents, began to show enlargement of the abdomen a few weeks after birth. This increased, and before death, which occurred at the age of six months, a hard nodular tumor mass was palpable in the upper abdomen. Death occurred apparently from general weakness. There was no family history of syphilis. The autopsy showed the greater part of the abdomen to be occupied by the liver, which weighed 943 grams, and measured 22 x 10 x 6 cm. The liver showed nodules throughout varying in size from that of a pea to that of a hen's egg, deep red in color, and slightly umbilicated. On section the nodules were composed of a dark-red centre surrounded by a white ring, and some showed white fibrous bands running in from the periphery. Microscopically the centres of the nodules were composed of cavities, or spaces, lined by endothelial cells and containing red and white blood cells. The capsule was composed of fibrous tissue and atrophic liver cells, and was somewhat infiltrated with lymphocytes. The parts of the liver distant from the tumor showed some disintegration and an increase of perilobular connective tissue.

A second case, of similar type, occurring in a child, aged seven years (first noticed at the age of five and one-half years), was reported by Sawyer.[6] In this case the family history was negative. The swelling of the abdomen was first noticed about a year and a half before death, and as it increased in size the child became emaciated and developed a slight fever. Autopsy showed no signs of congenital syphilis. Tuberculosis of the spleen, pancreas, and abdominal lymph nodes was present. The liver weighed about 900 grams, was nodular, and purplish in color. Microscopically, there was, throughout the liver, a widespread, nodular, angiomatous process. Between the nodules there was marked cirrhosis, with broad bands of fibrous tissue intersecting the liver and with prolongations surrounding the lobules. The diagnosis of angiomas with secondary cirrhosis was made.

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[1] Geschwülste in Kindersalte, 1905.

[2] Ibid.

[3] Archiv f. Kinderheilk., 1901, xxi, 291.

[4] Spezielle Path. Anat., 1907, p. 579

[5] Archiv. de Phys. norm et Path., 1885, vi, 553.

[6] Rep. Soc. Study Dis. Child, London, 1906, vi, 19.

The third case is described by Bruchanow.[7] It was a case of multiple angiomas of the liver in an infant, aged fifteen weeks, who showed also a number of angiomas of the skin. At autopsy an enlarged liver weighing 710 grams, and measuring 20 x 14 x 6 cm., was found, which contained a number of nodular tumors varying in size from that of a pea to that of a hen's egg. According to Bruchanow, these were true growing, cavernous angiomas. His diagnosis was subsequently attacked by Schmieden,[8] on grounds, however, which do not seem to us to be tenable. These 3 cases are the only ones resembling ours which we have been able to find in the literature.

Several theories have been offered to explain angiomas of the liver. For the majority of cases the old view that the tumor is the result of a capillary dilatation rather than a true blastoma is probably correct. Schmieden[9] and Adami[10] take this stand. Ribbert[11] and Bruchanow[12] explain the capillary dilatation as the result of a developmental defect, the bloodvessels having failed to establish normal relations with the liver cells. Pilliet[13] advocates a theory of fetal inclusion. Probably no one explanation will suffice for all cases. Our own case, as well as the 3 similar ones quoted from the literature, is probably more closely related to the tumors for which Mallory[14] has recently advocated the name "hemangio-endothelioma." These he defines as angiomas which show distinct proliferation of the endothelial cells, the essential cell of the bloodvessel, with distinct new formation of bloodvessels, a true blastoma, therefore. The term was applied by Mallory[15] particularly to those angiomas of the skin, which are not so uncommon, in which there is a true new formation of bloodvessels and which, as a rule, if recurrence is to be prevented, must be removed completely with a considerable portion of the surrounding tissue.

In our case the marked atrophy of the liver cells, the rapid increase in size of the liver, the young character of the fibrous tissue in the nodules, and, above all, the distinct evidence of endothelial cell proliferation, together with the clinical course of the case, all lead us to class the tumors as true blastomas, multiple congenital hemangio-endotheliomas. Specimens of the tissue were submitted to Professor Mallory, of Boston, and to Professors Pearce and Allen J. Smith, of Philadelphia, who have confirmed the diagnosis and to whom we wish to express our thanks.

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[7] Zeitsch. f. Heilk., 1899, xx, 131.

[8] Virchow's Archiv, 1900, clxi, 373.

[9] Loc. cit.

[10] Principles of Pathology, 1908, i, 751.

[11] Virchow's Archiv, 1898, cii, 351.

[12] Loc. cit.

[13] Le prog. méd., 1891, No. 29, p. 50.

[14] Jour. Exper. Med., 1908, x, 575.

[15] Jour. Amer. Med. Assoc., vol. lv, p. 1621.

OBSERVATIONS UPON THE PERSISTENCE OF GONOCOCCI IN THE MALE URETHRA.

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Surely none of us has any precise idea as to how long the gonococcus may survive in the glands that empty into the male urethra; for if they may survive a year or two, who shall deny them a decade? Not I, for one.

And yet I have not seen it. I have never known the gonococcus to survive in a male host for more than three years. I believe I could count upon my ten fingers the instances of a survival of eighteen months or more that have come under my observation. I recollect but one instance in which a year's treatment at my hands failed to free the urethra of gonococci. But I hasten to attribute this result to nature rather than to art. One of the most obstinate cases I ever undertook I treated for six months or more; he still had gonococci. He then deserted all treatment for six months. Then he married, and soon thereafter returned to show me that he was, so far as I could tell, entirely well. To be brief, I believe that the gonococcus does not persist in the male urethra for more than three years, while in at least 90 per cent. of cases it disappears, with or without treatment, within a year.

Upon what is this opinion founded? Not upon the cases and statistics to be cited. These do but illustrate and in some measure confirm my opinion. But this opinion is founded upon a double basis. First, my own inability, in spite of an alert interest in the subject for more than a decade, to find exceptions to the rule; and secondly, the insufficient evidence upon which exceptions are alleged either by physicians who have seen individual patients before they reached my hands, or by those who write.

What, then, is sufficient evidence that the male urethra is or is not free from gonococci?

We may divide the patients who come with this question upon their lips into four classes, as follows:

1. Patients who have apparently been cured at least three months of a gonorrhea and who show (*a*) no purulent shreds or free pus in the morning urine, and (*b*) no pus in the secretion expressed from prostate and vesicles. The observation of the